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T S13/5/ALL

13/5/1 (Item 1 from file: 340)
DIALOG(R)File 340:CLAIMS(R)/US Patent


10193957 2002-0137662 2002-0035621
**C/COMPOSITIONS AND METHODS FOR NEGATIVE REGULATION OF TGF-BETA PATHWAYS**
Document Type: Utility
Document Type: Patent Application-First Publication
Inventors: Laughon Allen S (US)
Assignee: Unassigned Or Assigned To Individual
Assignee Code: 68000

| | Publication | | | Application | |
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| | Kind | Number | Date | Number | Date |
| | A1 | US 20020137662 | 20020926 | US 2001810385 | 20010316 |
| Priority Applic: | | | | US 2001810385 | 20010316 |

Abstract: Methods for screening for compounds that are negative regulators of TGF- beta -regulated gene expression in mammalian cells are provided, including compositions identified therefrom.

Exemplary Claim: D R A W I N G

1. A method for identifying compounds that directly interact with a **Smad**protein or a Smad protein co-repressor to prevent protein-protein or protein-DNA interactions required for repression of **transcription**induced by TGF- beta , activin or bone morphogenetic protein signaling in cells comprising: (a) determining a first level of **transcription**detected in cells in the presence of a Smad protein and a CtBP protein before addition of a test compound; (b) contacting said cells with the test compound; and (c) determining a second level of **transcription**detected in cells in the presence of a Smad protein and a CtBP protein after addition of the test compound, wherein a decrease in the level of repression of**transcription**induced by the presence of the**Smad**protein and the CtBP protein is indicative of the ability of the test compound to interfere with**transcriptional** repression and to prevent repression of**transcription**that is produced by a TGF- beta , activin, or bone morphogenetic protein signal in cells.

Class: 514001000
Class Cross Ref: 435007200
IPC: A61K-031/00 (Edition 07)
IPC Cross Ref: G01N-033/53; G01N-033/567

13/5/2 (Item 1 from file: 349)
DIALOG(R)File 349:PCT FULLTEXT


00942700 **Image available**
**COMPOSITIONS AND METHODS FOR NEGATIVE REGULATION OF TGF-BETA PATHWAYS**
**COMPOSITIONS ET PROCEDES POUR LA REGULATION NEGATIVE DES VOIES DE FACTEUR DE CROISSANCE TRANSFORMANT BETA**
Patent Applicant/Assignee:
WISCONSIN ALUMNI RESEARCH FOUNDATION, 614 Walnut Street, Madison, WI 53705, US, US (Residence), US (Nationality)
Inventor(s):
LAUGHON Allen S, 717 Ottawa Trail, Madison, WI 53711, US,
Legal Representative:
LICATA Jane Massey (et al) (agent), Licata & Tyrrell P.C., 66 E. Main

Street, Marlton, NJ 08053, US,
Patent and Priority Information (Country, Number, Date):
Patent: WO 200276466 A1 20021003 (WO 0276466)
Application: WO 2002US8133 20020315 (PCT/WO US0208133)
Priority Application: US 2001810385 20010316
Designated States: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU
CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP
KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO
RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW
(EP) AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
(OA) BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
(AP) GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
(EA) AM AZ BY KG KZ MD RU TJ TM
Main International Patent Class: A61K-031/70
International Patent Class: A01N-043/04; C07K-014/00; C12Q-001/68
Publication Language: English
Filing Language: English
Fulltext Availability:
Detailed Description
Claims
Fulltext Word Count: 4842

English Abstract
Methods for screening for compounds that are negative regulators of TGF-beta-regulated gene expression in mammalian cells are provided, including compositions identified therefrom.

French Abstract
La presente invention concerne des procedes de criblage de composes qui sont des regulateurs negatifs de l'expression genetique regulee de TGF-beta dans des cellules mammaliennes, ainsi que des compositions qui sont identifiees a partir de ceux-ci. FIG. 1 : A beta-GALACTOSIDASE B 5 NANOGRAMMES DE MAD/20 NANOGRAMMES DE MEDEA C 0 NANOGRAMMES DE MEDEA, 0 NANOGRAMMES DE MAD D NANOGRAMMES DE LA PROTEINE dCtB

Legal Status (Type, Date, Text)
Publication 20021003 A1 With international search report.
Publication 20021003 A1 Before the expiration of the time limit for amending the claims and to be republished in the event of the receipt of amendments.
Examination 20030109 Request for preliminary examination prior to end of 19th month from priority date

**13/5/3 (Item 2 from file: 349)**
DIALOG(R)File 349:PCT FULLTEXT


00768498
**NUCLEIC ACID BINDING OF MULTI-ZINC FINGER** TRANSCRIPTION FACTORS
**LIAISON D'ACIDE NUCLEIQUE A DES FACTEURS DE** TRANSCRIPTION DE DOIGT **MULTI-ZINC**
Patent Applicant/Assignee:
VLAAMS INTERUNIVERSITAIR INSTITUUT VOOR BIOTECHNOLOGIE VZW, Rijvisschestraat 120, B-9052 Zwijnaarde, BE, BE (Residence), BE (Nationality), (For all designated states except: US)
Patent Applicant/Inventor:
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VERSCHUEREN Kristin, Twee Leeuwenstraat 22, B-3078 Everberg, BE, BE (Residence), BE (Nationality), (Designated only for: US)
REMACLE Jacques, Avenue des Lilas 7, B-4280 Hannut, BE, BE (Residence), BE (Nationality), (Designated only for: US)
Legal Representative:
VLAAMS INTERUNIVERSITAIR INSTITUUT VOOR BIOTECHNOLOGIE VZW (commercial

rep.), Rijvisschestraat 120, B-9052 Zwijnaarde, BE,
Patent and Priority Information (Country, Number, Date):
Patent: WO 200100864 A2-A3 20010104 (WO 0100864)
Application: WO 2000EP5582 20000609 (PCT/WO EP0005582)
Priority Application: EP 99202068 19990625
Designated States: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
(EP) AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
(OA) BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
(AP) GH GM KE LS MW MZ SD SL SZ TZ UG ZW
(EA) AM AZ BY KG KZ MD RU TJ TM
Main International Patent Class: C12Q-001/00
Publication Language: English
Filing Language: English
Fulltext Availability:
Detailed Description
Claims
Fulltext Word Count: 23477

English Abstract
The invention concerns a method of identifying**transcription**factors comprising providing cells with a nucleic acid sequence at least comprising a sequence CACCT as bait for the screening of a library encoding potential**transcription**factors and performing a specificity test to isolate said factors. Preferably the bait comprises twice the CACCT sequence, more particularly the bait comprises one of the sequences CACCT-N-CACCT, CACCT-N-AGGTG, AGGTG-N-CACCT or AGGTG-N-AGGTG wherein N is a spacer sequence. The identified**transcription**factor(s) using the method according to the invention comprises separated clusters of zinc fingers such as for example a two-handed zinc finger**transcription** factor. The present invention further discloses that at least one such zinc finger**transcription**factor, denominated as SIP1 , induces tumor metastasis by downregulation of the expression of E-cadherin. Compounds interfering with**SIP1**activity can thus be used to prevent tumor invasion and metastasis.

French Abstract
L'invention concerne un procede d'identification de facteurs de **transcription**permettant d'apporter a des cellules une sequence d'acide nucleique comprenant au moins une sequence CACCT comme appat dans le criblage d'une bibliotheque codant les facteurs de**transcription** potentiels et executant un test specifique pour eliminer ces facteurs. L'appat comprend, de preference, deux sequence CACCT, plus particulierement, l'appat comprend une des sequences CACCT-N-CACCT, CACCT-N-AGGTG, AGGTG-N-CACCT ou AGGTG-N-AGGTG dans lesquelles N represente une sequence d'espacement. Le(s) facteur(s) de**transcription** identifie(s) utilisant le procede de l'invention comprend (comprennent) des groupes separes de doigts de zinc tels que par exemple un facteur de **transcription**de doigt de zinc a deux mains. En outre, la presente invention indique qu'au moins un facteur de**transcription**de doigt de zinc, appele**SIP1** , provoque des metastases tumorales par regulation a la baisse de l'expression de E-cadherine. Les composes interferants avec l'activite**SIP1**peuvent ainsi etre utilises pour empecher l'invasion de tumeurs et la metastase.

Legal Status (Type, Date, Text)
Publication 20010104 A2 Without international search report and to be republished upon receipt of that report.
Examination 20010308 Request for preliminary examination prior to end of 19th month from priority date
Search Rpt 20011129 Late publication of international search report
Republication 20011129 A3 With international search report.
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T S30/5/ALL

30/5/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


13094321 BIOSIS NO.: 200100301470
**The corepressorCTBP is involved in Evi-1 mediated repression of TGF-beta signaling.**
AUTHOR: Izutsu Koji(a); Kurokawa Mineo(a); Imai Yoichi(a); Mitani Kinuko(a); Hirai Hisamaru(a)
AUTHOR ADDRESS: (a)Department of Hematology and Oncology, Graduate School of Medicine, University of Tokyo, Tokyo**Japan
JOURNAL: Blood 96 (11 Part 1):p90a November 16, 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000
SPONSOR: American Society of Hematology
ISSN: 0006-4971
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Evi-1 is a zinc finger nuclear protein whose inappropriate expression leads to leukemic transformation of hematopoietic cells in mice and humans. Evi-1 is shown to be highly expressed in human myeloid leukemias and myelodysplastic syndromes by chromosomal rearrangements involving 3q26. It is also aberrantly expressed as a fusion transcript with AML1 (AML1/Evi-1), which leads to blastic transformation in patients with chronic myelogenous leukemia. We previously showed that Evi-1 and AML1/Evi-1 block the antiproliferative effect of TGF-beta. They represses TGF-beta signaling by direct interaction with Smad3 through their first zinc finger motif. Here, we demonstrate that Evi-1 represses Smad-induced **transcription**by recruiting CtBP as a corepressor. CtBP was originally identified as a protein which interacts with C-terminal region of adenoviral oncoprotein E1A.**CtBP**is ubiquitously expressed including hematopoietic cells, and has been shown to act as a corepressor of certain transcriptional repressors, such as BKLF, FOG, and TCF. We show that Evi-1 directly associates with CtBP1 through one of the consensus binding motifs, and this association is required for efficient inhibition of TGF-beta signaling. A specific inhibitor for histone deacetylase (HDAc) alleviates Evi-1-mediated repression of TGF-beta signaling, suggesting that HDAc is involved in the transcriptional repression by Evi-1. This identifies a novel function of Evi-1 as a member of corepressor complexes and suggests that aberrant recruitment of corepressors is one of the mechanisms for Evi-1-induced leukemogenesis.

REGISTRY NUMBERS: 115640-43-2:**EVI-1**
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Tumor Biology
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae)
ORGANISMS: PARTS ETC: chromosome 3--q26
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Primates; Vertebrates
DISEASES: acute myeloid leukemia--blood and lymphatic disease, neoplastic disease
CHEMICALS & BIOCHEMICALS: AML1/Evi-1; **CTBP** --corepressor; Evi-1 -- zinc finger nuclear protein; transforming growth factor-beta--EVI-1 mediated signaling repression
MISCELLANEOUS TERMS: Meeting Abstract
ALTERNATE INDEXING: Leukemia, Myeloid (MeSH)
CONCEPT CODES:

24010 Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10060 Biochemical Studies-General
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
15002 Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
15006 Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects

BIOSYSTEMATIC CODES:
86215 Hominidae

**30/5/2 (Item 2 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


13037417 BIOSIS NO.: 200100244566
**The corepressor**CtBP interacts with Evi-1 to repress transforming growth **factor beta signaling.**
AUTHOR: Izutsu Koji; Kurokawa Mineo; Imai Yoichi; Maki Kazuhiro; Mitani Kinuko; Hirai Hisamaru(a)
AUTHOR ADDRESS: (a)Dept of Hematology and Oncology, Graduate School of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo, 113-8655: hhirai-tky@umin.ac.jp**Japan
JOURNAL: Blood 97 (9):p2815-2822 May 1, 2001
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Evi-1 is a zinc finger nuclear protein whose inappropriate expression leads to leukemic transformation of hematopoietic cells in mice and humans. This was previously shown to block the antiproliferative effect of transforming growth factor beta (TGF-beta). Evi-1 represses TGF-beta signaling by direct interaction with Smad3 through its first zinc finger motif. Here, it is demonstrated that Evi-1 represses Smad-induced**transcription**by recruiting C-terminal binding protein ( **CtBP** ) as a corepressor. Evi-1 associates with CtBP1 through one of the consensus binding motifs, and this association is required for efficient inhibition of TGF-beta signaling. A specific inhibitor for histone deacetylase (HDAc) alleviates Evi-1-mediated repression of TGF-beta signaling, suggesting that HDAc is involved in the transcriptional repression by Evi-1. This identifies a novel function of Evi-1 as a member of corepressor complexes and suggests that aberrant recruitment of corepressors is one of the mechanisms for Evi-1-induced leukemogenesis.

REGISTRY NUMBERS: 115640-43-2:**EVI-1** ; 9076-57-7: HISTONE DEACETYLASE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Tumor Biology
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae); mouse (Muridae)--animal model
ORGANISMS: PARTS ETC: hematopoietic cells--blood and lymphatics
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Rodents; Vertebrates
DISEASES: myelodysplastic syndrome--blood and lymphatic disease,

neoplastic disease
CHEMICALS & BIOCHEMICALS: C-terminal binding protein 1 {CtBP1}--corepressor; **Evi-1** --zinc finger nuclear protein; histone deacetylase; transforming growth factor-beta--signaling repression
MISCELLANEOUS TERMS: leukemogenesis
ALTERNATE INDEXING: Myelodysplastic Syndromes (MeSH)
CONCEPT CODES:
10060 Biochemical Studies-General
02506 Cytology and Cytochemistry-Animal
02508 Cytology and Cytochemistry-Human
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
15002 Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
15006 Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects
24010 Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

**30/5/3 (Item 3 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


12248967 BIOSIS NO.: 200000002469
**Basic Kruppel-like factor functions within a network of interacting haematopoietic**transcription factors.
AUTHOR: Turner Jeremy; Crossley Merlin(a)
AUTHOR ADDRESS: (a)Department of Biochemistry, G08, University of Sydney, Sydney, NSW, 2006**Australia
JOURNAL: International Journal of Biochemistry & Cell Biology 31 (10):p 1169-1174 Oct., 1999
ISSN: 1357-2725
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Basic Kruppel-like Factor (BKLF) is a recently recognized member of a small group of**transcription**factors that bind CACCC motifs in DNA, by means of three highly conserved C-terminal Kruppel-like (typically Cys-X2-4-Cys-X12-His-X3-4-His) zinc fingers. Together with Erythroid Kruppel-like Factor (EKLF), it is one of the most abundant CACCC-binding proteins in erythroid cells. In contrast to EKLF, BKLF can act to repress**transcription**and thus may serve to moderate EKLF activity in vivo. Interestingly, it has also been shown that BKLF expression in erythroid cells is dependent on EKLF. Analysis of proteins interacting with BKLF indicates that it represses**transcription**by recruiting the general co-repressor protein**CtBP** , a cofactor that also associates with other haematopoietic transcriptional repressors such as Evi-1 and ZEB/AREB6. The observation that mice deficient in BKLF exhibit a myeloproliferative disorder suggests that BKLF regulates important processes involved in haema topoietic differentiation.

REGISTRY NUMBERS: 115640-43-2:**EVI-1**
DESCRIPTORS:
MAJOR CONCEPTS: Molecular Genetics (Biochemistry and Molecular Biophysics); Blood and Lymphatics (Transport and Circulation)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia

ORGANISMS: human (Hominidae); mouse (Muridae)
ORGANISMS: PARTS ETC: erythroid cell--blood and lymphatics
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Rodents; Vertebrates
DISEASES: myeloproliferative disorder--blood and lymphatic disease
CHEMICALS & BIOCHEMICALS: **CtBP** ; DNA-- transcription ; Evi-1 ; ZEB/AREB6; basic Kruppel-like factor--**transcription**factor; erythroid Kruppel-like factor--**transcription**factor
CONCEPT CODES:
10060 Biochemical Studies-General
02506 Cytology and Cytochemistry-Animal
10300 Replication, Transcription, Translation
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
03506 Genetics and Cytogenetics-Animal
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

**30/5/4 (Item 4 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


11661557 BIOSIS NO.: 199800443288
**Cloning and characterization of mCtBP2, a co-repressor that associates with basic Kruppel-like factor and other mammalian transcriptional regulators.**
AUTHOR: Turner Jeremy; Crossley Merlin(a)
AUTHOR ADDRESS: (a)Dep. Biochem., G08, Univ. Sydney, Sydney, NSW 2006** Australia
JOURNAL: EMBO (European Molecular Biology Organization) Journal 17 (17):p 5129-5140 Sept. 1, 1998
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Basic Kruppel-like factor (BKLF) is a zinc finger protein that recognizes CACCC elements in DNA. It is expressed highly in erythroid tissues, the brain and other selected cell types. We have studied the activity of BKLF and found that it is capable of repressing **transcription** , and have mapped its repression domain to the N-terminus. We carried out a two-hybrid screen against BKLF and isolated a novel clone encoding murine C-terminal-binding protein 2 (mCtBP2). mCtBP2 is related to human**CtBP** , a cellular protein which binds to a Pro-X-Asp-Leu-Ser motif in the C-terminus of the adenoviral oncoprotein, E1a. We show that mCtBP2 recognizes a related motif in the minimal repression domain of BKLF, and the integrity of this motif is required for repression activity. Moreover, when tethered to a promoter by a heterologous DNA-binding domain, mCtBP2 functions as a potent repressor. Finally, we demonstrate that mCtBP2 also interacts with the mammalian **transcription**factors Evi-1, AREB6, ZEB and FOG. These results establish a new member of the**CtBP**family, mCtBP2, as a mammalian co-repressor targeting diverse transcriptional regulators.

REGISTRY NUMBERS: 115640-43-2:**EVI-1**
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Methods and Techniques
BIOSYSTEMATIC NAMES: Diptera--Insecta, Arthropoda, Invertebrata, Animalia ; Mammalia--Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia , Vertebrata, Chordata, Animalia
ORGANISMS: mammal (Mammalia); NIH 3T3 (Muridae); SL2 (Diptera)

BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Arthropods; Chordates ; Insects; Invertebrates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates

CHEMICALS & BIOCHEMICALS: basic Kruppel-like factor; mCtBP2 {murine C-terminal-binding protein 2}--characterization, cloning; transcriptional regulators; AREB6--**transcription**factor; DNA; **Evi-1** -- transcription factor; FOG-- transcription factor; ZEB--**transcription**factor

METHODS & EQUIPMENT: cloning--Recombinant DNA Technology, cloning method; gel mobility shift assay--Analysis/Characterization Techniques--CB, analytical method; gel retardation assay--Analysis/Characterization Techniques--CB, analytical method; transactivation assay--Analysis/Characterization Techniques--CB, analytical method; transrepression assay--Analysis/Characterization Techniques--CB, analytical method; two-hybrid screen--Qualitative/Quantitative Techniques, screening method

MISCELLANEOUS TERMS: amino acid sequence

CONCEPT CODES:
10060 Biochemical Studies-General
03506 Genetics and Cytogenetics-Animal
10050 Biochemical Methods-General

BIOSYSTEMATIC CODES:
75314 Diptera
85700 Mammalia-Unspecified
86375 Muridae

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